PAPER

Deglutitive laryngeal closure in stroke patients

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Background: Dysphagia has been reported in up to 70% of patients with stroke, predisposing them to aspiration and pneumonia. Despite this, the mechanism for aspiration remains unclear.
Aims: To determine the relationship between bolus flow and laryngeal closure during swallowing in patients with stroke and to examine the sensorimotor mechanisms leading to aspiration.
Methods: Measures of swallowing and bolus flow were taken from digital videofluoroscopic images in 90 patients with stroke and 50 healthy adults, after repeated volitional swallows of controlled volumes of thin liquid. Aspiration was assessed using a validated Penetration–Aspiration Scale. Oral sensation was also measured by electrical stimulation at the faucial pillars.
Results: After stroke, laryngeal ascent was delayed (mean (standard deviation (SD)) 0.31 (0.06) s, $p<0.001$), resulting in prolongation of pharyngeal transit time (1.17 (0.07) s, $p<0.001$) without a concomitant increase in laryngeal closure duration (0.84 (0.04) s, $p=0.9$). The delay in laryngeal elevation correlated with both the severity of aspiration ($r=0.5$, $p<0.001$) and oral sensation ($r=0.5$, $p<0.001$).
Conclusions: After stroke, duration of laryngeal delay and degree of sensory deficit are associated with the severity of aspiration. These findings indicate a role for sensorimotor interactions in control of swallowing and have implications for the assessment and management of dysphagia after stroke.

Dysphagia has been reported in up to 70% of patients with stroke, with the major clinical complication being deglutitive aspiration.[1] Historically, the prevailing belief has been that for most patients, aspiration resolves spontaneously within days.[2] In fact, aspiration is now known to persist well beyond 6 months in up to 50% of patients with stroke,[3] and the incidence of pneumonia remains unacceptably high.[4] Despite this, objective measurements of relationships between bolus flow and laryngeal closure, factors that might predict patients at risk of aspiration, have never been systematically determined in a cohort of patients with pure stroke.

Patients with hemispheric stroke are described as presenting with "difficulty initiating coordinated movement",[5] "delayed pharyngeal response"[6] and "delayed or absent swallow reflex".[7] It has been speculated that these features are associated with a breakdown in the descending efferent outputs from the cortex, which initiate the brain stem-mediated pharyngeal response and laryngeal protection.[8] However, the neural control of swallowing integrates peripheral afferent inputs from the oropharynx with descending efferent outputs.[9] Recent work by Aviv *et al*[10] has suggested that patients with stroke and dysphagia also have impairments in pharyngeal sensation. Despite these observations, whether such sensory deficits specifically affect deglutitive laryngeal function remains unknown.

The protective mechanism that guards against aspiration is laryngeal closure, which is achieved by a sequence of events, beginning with cessation of respiration, approximation of the arytenoids and adduction of the vocal cords. Complete closure is achieved by a combination of laryngeal ascent and epiglottic descent.[11 12] Laryngeal closure duration (LCD) modulates to accommodate bolus characteristics[13]; however, whether the timing of laryngeal ascent is fixed or responsive to bolus characteristics is not known.[14 15]

Studies on swallowing in humans have implicated the faucial pillar, an area innervated by the glossopharyngeal nerve, as a "trigger" for the onset of laryngeal ascent.[14 16 17] Studies on patients with aspirating stroke have suggested that delayed onset of laryngeal ascent can be shortened by stimulation at the faucial pillar.[18] Stimulation at the faucial pillar has been considered beneficial because it stimulates mechanoreceptors at the head of the hypopharynx, which relay information to the brain stem and cortex to initiate laryngeal closure. The evidence to support this contention is, however, controversial, with some authors suggesting that higher cortical functions are more important.[19] Furthermore, some studies have suggested that the important receptive sensory fields for triggering swallowing do not include the faucial pillar region.[20] Thus, although the faucial pillar is the anatomical gateway to the hypopharynx, it may have no role in the onset of laryngeal ascent. A systematic examination of this relationship in health and stroke is, however, lacking.

The aims of this study were therefore to examine the temporal linkage of bolus transit and laryngeal closure in healthy adults and to compare our findings with those obtained from patients after hemispheric stroke. We hypothesised that in healthy participants, both the initiation and duration of laryngeal closure would be modulated by sensory input, and that this relationship would be impaired in patients with stroke. We also postulated that altered oral sensation would relate to the severity of aspiration in patients with stroke.

METHODS

Participants

In all, 140 people took part in the studies (50 healthy participants, 29 men, mean age 38 years, in two age groups <55 (n = 38) and >55 years (n = 11) and 90 participants with hemispheric stroke, 53 men, mean age 69 years, range 29–92 years). Healthy participants were recruited from the local community. Patients with stroke were recruited from a large teaching hospital and studied within 2 weeks of stroke (mean interval 10 days, range 6–13 days). Participants were excluded if they had a history of difficulty in swallowing, neurological disease, intercurrent illness or upper gastrointestinal disease.

Abbreviations: LCD, laryngeal closure duration; OLE, onset of laryngeal elevation; OTT, oral transit time; PAS, Penetration–Aspiration Scale; PTT, pharyngeal transit time; SRT, swallow response time

They were also ineligible if they were unable either to give informed consent or unable to participate in the videofluoroscopy examination. All study protocols were presented to and approved by the local research ethics committee, and all participants gave written consent before the study began.

Assessment of damage caused by stroke

In all patients, computerised tomography scans of the brain were used to confirm the type, size and site of the lesion. All scans were analysed by a neuroradiologist, blinded to the patient's clinical presentation and swallowing status. Lesions were characterised according to their type (ischaemic *v* haemorrhagic), side (left or right) and volume, using the Alberta Stroke Programme Early CT Score.[21] The 100-point Barthel Index was recorded as an indication of functional impairment.[22]

Assessment of swallowing

All participants were assessed by videofluoroscopy using barium liquid (60% wt/vol, EZ-HD, E-Z-EM, London, UK). Images were acquired (Siemens Fluorospot H SIRESKOP SX Unit, Siemens Aktiengesellschaft Medical Engineering, Erlangen, Germany) in real time using continuous fluoroscopy at 30 frames/s (Videomed DI TV system) and recorded by a digital video at 25 frames/s (Sony DHR 1000, Sony UK, Weybridge, Surrey, UK). Lateral images of the oropharynx were subsequently acquired without magnification, according to previously described protocols.[23] The total screening time was kept below 80 s (range 42–73 s) in all cases, giving a radiation dose of <0.3 mSv.

Videofluoroscopic measures

Two measures of bolus transit were chosen to evaluate bolus flow through the mouth: oral transit time (OTT) and pharyngeal transit time (PTT). In addition, measures of time from arrival of the bolus head at the hypopharynx (swallow response time, SRT) to onset of laryngeal elevation (OLE) and duration of laryngeal closure (LCD) were recorded.

Definition of terms

OTT was defined as the interval, in seconds, between the first frame showing elevation of the tongue tip (with subsequent posterior movement of the bolus tail) and the first frame showing the arrival of the head of the bolus at the hypopharynx (defined as the anatomical point where the ramus of the mandible crosses the tongue base).

PTT was defined as the interval, in seconds, between the first frame showing the arrival of the bolus head at the hypopharynx to the last frame showing the tail of the bolus passing through the mid-margin of the upper oesophageal sphincter.

SRT was defined as the interval, in seconds, between the first frame showing the arrival of the bolus head at the hypopharynx to the first frame showing upward excursion of the larynx.

LCD was defined as the interval, in seconds, between the first frame showing contact between the inferior surface of the epiglottis and arytenoids and the first frame showing that contact had ceased.

Temporal reference points

In addition to the primary measures of bolus transit and laryngeal closure, in all healthy participants and 52 patients with stroke, the temporal relationship between the elevation of the tongue tip (swallow onset) and laryngeal closure was obtained by referencing three additional time points: the OLE, the time to complete laryngeal elevation and the time to laryngeal reopening, with the first frame denoting the onset of elevation of the tongue tip (time = 0).

Laryngeal penetration and aspiration were assessed for all swallows using a previously developed and validated 8-point Penetration–Aspiration Scale (PAS), which describes the severity of airway compromise.[24] Given that normal participants are known to score 1–2 on the PAS,[25] patients were considered to have abnormal laryngeal protection (aspirators) if they scored $\geqslant 3$ on one or more swallows on the PAS. Their worst aspiration score was used to determine the relationship with demographic and swallowing measures.

Assessment of sensation of the faucial pillar

To provide an objective and reliable measure of oral sensation, the sensitivity of the faucial pillar was assessed using a 2-mm fingertip electrode (St Mark's Pudendal Electrode, Medtronic Diagnostics A/S, Tonsbakken, Skovlunde, Denmark) placed digitally on to each anterior faucial pillar. The electrode was connected to a constant current stimulator (Model DS7, Digitimer, Welwyn Garden City, Herts, UK), and the stimuli were delivered via a trigger generator (Model DG2, Digitimer) at a frequency of 5 Hz (square wave duration 200 μs). For each faucial pillar, the intensity of the stimulus was increased in a stepwise fashion in increments of 0.2 mA from zero until the participant just perceived the electrical sensation. This process was repeated, across the right and left faucial pillars, three stimulations per side, in random order.

EXPERIMENTAL PROTOCOLS

Protocol 1: Pharyngeal response patterns in health

In all, 38 healthy participants were assessed by videofluoroscopy using liquid boluses (6×5 ml). All participants were asked to retain the liquid in their mouth until instructed to swallow.

Protocol 2: Sensory thresholds and the effect of bolus volume in health

Sensation of the faucial pillar was measured in 12 healthy participants (6 men, mean age 38 years, range 28–60 years) after protocol 1 to determine the normal range of sensation of the faucial pillar.

A further 12 healthy participants (10 men, mean age 39 years, range 24–55 years) were also assessed using boluses of 5, 10, 15 and 20 ml of barium to determine the effect of bolus size on the initiation and duration of laryngeal closure.

Protocol 3: Pharyngeal response after cortical hemispheric stroke

In addition to the measures of bolus flow and laryngeal closure described above, airway compromise was evaluated for all swallows using the PAS for the 90 stroke patients.[19]

Protocol 4: Sensory thresholds and the effect of bolus volume in stroke

Sensation of the faucial pillar was measured in 41 patients recruited to protocol 3.

Ten patients from protocol 3 were assessed using multiple bolus volumes of 5, 10, 15 and 20 ml of barium to determine the effect of bolus size on the initiation and duration of laryngeal closure.

DATA ANALYSIS

All data are expressed as mean (standard error of mean). A p value <0.05 was taken to indicate significance unless otherwise stated. Descriptive and comparative analyses were made using SPSS software V.11.1. Comparison of the performance within and between participants was made using analysis of variance. Interparticipant comparisons were made using individual means for each measure. Spearman's correlation coefficients were used to explore the relationships between age, videofluoroscopy timings and severity of aspiration.

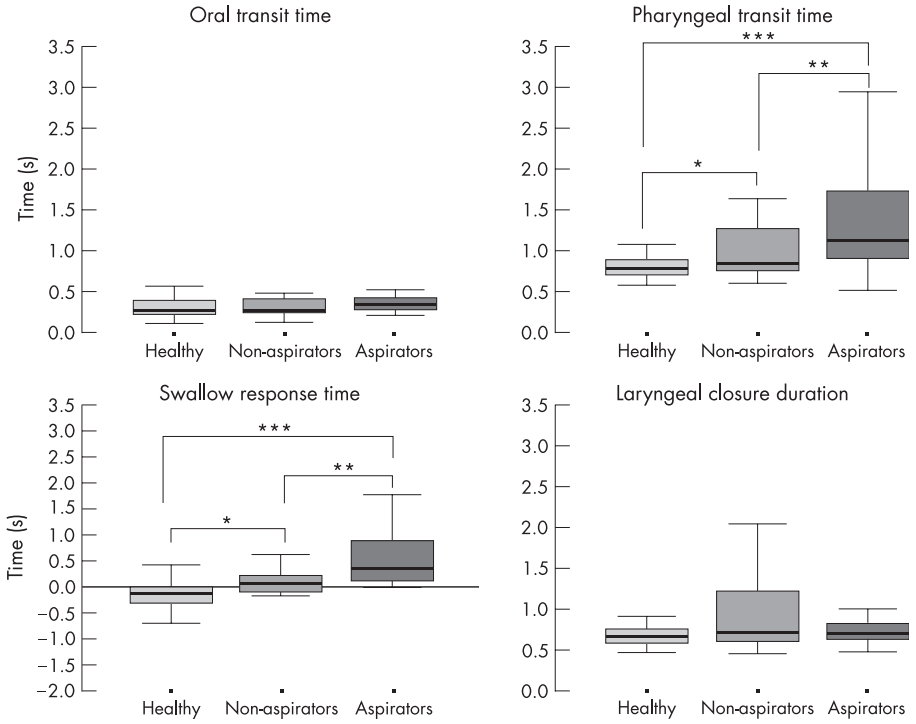

Figure 1 Compared with the healthy participants who had anticipatory laryngeal closure, swallow response time was delayed after stroke (***p = 0.005) in both non-aspirating (*p = 0.05) and aspirating patients (**p = 0.001). Pharyngeal transit time was also prolonged after stroke (***p = 0.001) in both the non-aspirating (*p = 0.001) and aspirating (**p = 0.001) patients. Oral transit time and laryngeal closure duration remained similar in the three groups. Data are median (range).

RESULTS

Health

Laryngeal vestibule closure patterns

The onset of laryngeal ascent preceded arrival of the bolus at the hypopharynx, and therefore swallow response time had a negative value (mean (standard deviation (SD)) −0.18 (0.04); fig 1). Participants with the longest PTT also had the longest LCD (r = 0.4, p = 0.02).

Oral sensation

The 95% confidence interval (CI) for faucial pillar threshold was 1.52 to 2.97 mA and there was no correlation between age of participant and sensory threshold (r = −0.13, p = 0.7). Thresholds were similar for the right (mean (SD) 2.11 (0.27) mA) and left (2.41 (0.40) mA) faucial pillar and were consistent within participants with high intraclass correlation coefficients between three stimulations on the right (r = 0.9, p = 0.9) and left (r = 0.9, p = 0.4).

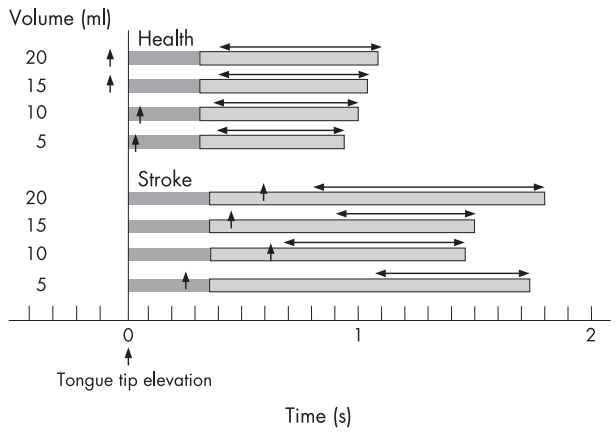

Figure 2 Schematic illustration of bolus volume showing mean values of data. The onset (vertical arrow) and duration (horizontal arrow) of laryngeal closure in health and stroke are shown. Time = 0 marks the first videofluoroscopic frame showing elevation of the tongue tip. The horizontal shading represents the time taken for the bolus to travel through the mouth (dark grey) and pharynx (light grey) and ends with the last frame showing passage of the bolus through the cricopharynx. The arrival of the bolus head at the hypopharynx is the point at which the light and grey bars meet.

The effect of bolus volume on laryngeal closure

As bolus volume increased, there was a more rapid transfer of the bolus through the mouth—that is, reduced OTT (p = 0.02), the onset of laryngeal ascent still occurred before the arrival of the bolus head at the hypopharynx, even with the largest bolus volumes, and SRT was unaffected (p = 0.7). By contrast, LCD increased systematically as bolus volume increased (p = 0.009; fig 2).

Stroke

Patient characteristics

Of the 90 patients with stroke studied, 47 (28 men, mean age 74 years, range 48–92 years) aspirated on videofluoroscopy (VF) (table 1). Aspirating patients were older (mean age (SD) 74 (1.3) years *v* 65 (1.7) years, p<0.001) and had larger lesion volumes (p = 0.04) than non-aspirators. Sex distribution, affected hemisphere, disability (Barthel Index) and stroke type (ischaemic *v* haemorrhagic) were similar in the two groups.

Laryngeal closure patterns

The larynx was lowered, in its resting position, when the bolus head entered the hypopharynx. Once triggered, the interval between the OLE and complete laryngeal closure was similar to healthy participants. LCD was consistent across swallows, but unlike healthy participants, there was no relationship between LCD and PTT (r = 0.23, p = 0.19).

The effect of bolus volume on laryngeal closure

The patients with stroke behaved similarly to the healthy participants, in that increasing bolus volume shortened OTT (p = 0.03), and PTT was unaffected. In contrast with healthy

Table 1 Demographics of patients with stroke

	Non-aspirators (n = 43)	Aspirators (n = 47)	p Value
Mean (SD) age (years)	65 (1.7)	74 (1.3)	<0.001
Sex	25 men 18 women	28 men 19 women	
Barthel	49 (5)	40 (4)	0.2
Hemisphere*	16 right, 18 left	19 right, 21 left	
Type	31 ischaemic 3 haemorrhagic	35 ischaemic 5 haemorrhagic	
Lesion volume (ASPECTS)	2.36 (0.3)	3.0 (0.3)	0.04

ASPECTS, Alberta Stroke Programme Early CT Score.
*In all, 16 patients (7 aspirators) had early computed tomography scans that were normal.

volunteers, the onset of laryngeal ascent always occurred after arrival of the bolus head at the hypopharynx and LCD did not increase as bolus volume increased ($p = 0.25$; fig 2).

Laryngeal closure patterns and aspiration

We found a relationship between SRT and severity of aspiration score ($r = 0.5$, $p<0.001$; fig 3). SRT was more delayed in aspirating patients (mean (SD) 0.53 (0.09) *v* 0.06 (0.06), $p<0.001$) and therefore PTT was prolonged (1.45 (0.10) *v* 0.87 (0.05), $p<0.001$; fig 1). However, OTT and LCD remained similar in the non-aspirating and aspirating patients, respectively, (0.31 (0.04) *v* 0.36 (0.04)) and (0.85 (0.07) *v* 0.83 (0.05), $p = 0.9$).

Oral sensation in stroke

The 95% CI for faucial pillar sensory threshold was 1.92 to 3.91 mA for the non-aspirators and 3.1 to 4.5 mA for the aspirating patients. We found no relationship between sensation of the faucial pillar and age ($r = 0.05$, $p = 0.7$) or lesion volume ($r = -0.07$, $p = 0.7$) in either the non-aspirating or aspirating patients. Of the 41 patients with stroke, 27 had sensory thresholds outside the (healthy) normal range (95% CI 1.54 to 2.97 mA). We also found a positive correlation between sensation of the faucial pillar and SRT ($r = 0.5$, $p = 0.001$). In 11 patients, the sensory thresholds fell outside the 95% CI of both the normal participants and patients with non-aspirating stroke (fig 4). These patients showed a fourfold increase in laryngeal delay (odds ratio 4.3, CI 2.2 to 4.8, $p = 0.03$), reduced LCD ($r = -0.4$, $p = 0.05$), and were all aspirators. As with the healthy participants, sensory thresholds were consistent in participants with high intraclass correlation coefficients (0.9, $p = 0.22$) between three stimulations. Thresholds were similar for the right (3.84 (0.4) mA) and left (3.36 (0.25) mA) faucial pillars in both right and left hemispheric stroke.

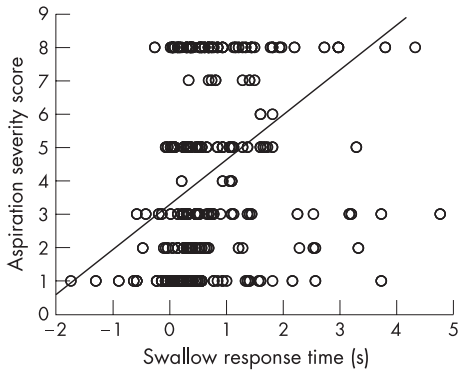

Figure 3 After stroke, there is evidence of a relationship between the onset of laryngeal ascent (swallow response time) and severity of aspiration. Data of all swallows performed by patients with stroke for 5 ml liquid bolus are presented.

DISCUSSION

This study has shown that, in health, the onset of laryngeal ascent consistently precedes arrival of the bolus at the hypopharynx. This contrasts with previous studies, which suggest that the arrival of the bolus at the hypopharynx triggers the onset of laryngeal ascent,[26 27] but concurs with more recent studies[28–31] that report anticipatory laryngeal ascent as a feature of normal swallowing. Our study shows that not only is early ascent lost after stroke but also that patients with the longest delays have the most severe aspiration. The findings of this study advance our knowledge on the onset of laryngeal ascent for liquid boluses in a homogeneous population of patients with hemispheric stroke and add to the body of work in health[32] and neurological disease.[33] These measurements are important for assessment and evaluation of the effect of treatment, and therefore warrant further discussion.

We experimented with intra-bolus stimuli and oral sensory thresholds to explore the role of sensation in the onset of laryngeal ascent. Previous studies have suggested that the interval between the arrival of the bolus at the hypopharynx and laryngeal ascent can be shortened by increasing bolus volume and that this might offer a therapeutic strategy for improving function.[34 35] However, we found no evidence to support this notion, as the onset of laryngeal ascent remained constant irrespective of bolus volume. Furthermore, patients with stroke having delayed onset of laryngeal ascent did not show the expected (normal) response with larger volumes reported in other studies. Our findings are consistent with experiments on healthy people which show timely laryngeal ascent despite removal of sensation by oropharyngeal anaesthesia,[20] and challenge the use of bolus volume as a technique to accelerate the onset of laryngeal ascent.

We were also interested in determining relationships between sensation of the faucial pillar and the onset of laryngeal ascent. All but four patients had abnormal onset of laryngeal ascent; however, most were able to perceive a stimulus at the faucial pillar, albeit at greater intensity than the healthy participants. Moreover, it was interesting that sensation of the faucial pillar, while reduced, was symmetrically reduced between the two sides. This might imply that (unilateral) stroke can alter sensation from both sides of the oral cavity. In support of this notion, cerebral evoked potentials (CEP) to stimulation at the anterior faucial pillar were found to project to both hemispheres with ipsilateral cortical predominance.[36] It is therefore conceivable that a unilateral stroke may

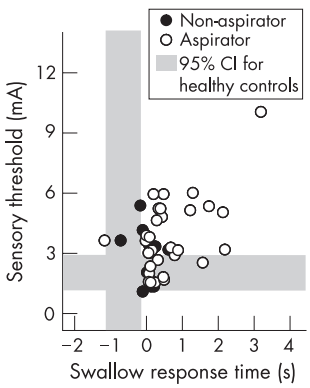

Figure 4 Relationship between sensation of the faucial pillar and swallow response time (SRT). All but one patient with stroke had an SRT outside the 95% CI for healthy controls. Twenty seven patients also had sensory thresholds outside the 95% CI for healthy controls. Aspirators had more prolonged SRT and increased sensory threshold than non-aspirating patients.

effect both the ipsilateral and contralateral ascending projections from each faucial pillar, thus altering sensitivity. Regardless of mechanism, and although we did not assess sensory function in the entire upper aerodigestive tract, these data offer two important messages. Firstly, hemispheric stroke disrupts the timing of laryngeal ascent even where oral sensation is intact. Secondly, patients with severe impairments in sensation of the faucial pillar have the longest delays and always aspirate.

From this study, it would be too speculative to extrapolate the data on faucial pillars to global sensory abnormalities in stroke. Indeed, it would also be premature to conclude that the reduction in sensation is related to the pathology of stroke, or that abnormal stimulus perception occurs at lower or higher processing levels in the afferent pathway. It remains controversial whether the diminished sensation of the faucial pillars is causal with respect to delay in swallowing. These findings, nonetheless, do suggest that after stroke, in contrast with health, sensory feedback from the faucial pillars may be important in modulating the onset of laryngeal ascent. By way of explanation, it is possible to speculate that patients with stroke and damaged motor control become more reliant on sensory cues and thus additional sensory deficits, of any aetiology, limit the adaptive process in a manner not seen in healthy participants. Our data add to existing work, which suggests a relationship between supraglottic and sensory abnormalities in patients with stroke and dysphagia[37] by providing a detailed account of the relationship between sensation of the faucial pillar and laryngeal function. Our data may also help to explain the variable response to sensory interventions,[38–40] as patients with abnormal sensation might be expected to respond differently from those with normal sensation. Our data indicate the need for a thorough assessment of sensation before recruitment to intervention studies.

An important further consideration is what other contributory factors influence aspiration, as we identified a small subgroup of patients who, despite having normal laryngeal ascent and sensation, still aspirated. It is possible that in some patients, increased laryngeal penetration could occur as a consequence of age-related changes to the bony framework, ligament structures of the larynx or viscoelastic properties of the epiglottis, larynx and pharyngeal body. It is also important to note that one of the limitations of videofluoroscopy is that it is difficult to visualise the vocal cords. As it is recognised that cord closure precedes laryngeal movement and forms an important component of airway protection, further information from fibre-optic evaluation of swallowing could add clarity to the mechanism of aspiration in this group.

We conclude that in health, the OLE is tightly synchronised with bolus transit, and likely to be dependent on higher centres in the central nervous system. The primary swallow abnormality in stroke seems to be a delay in the OLE; the longer the delay, the more severe the aspiration. In addition, patients with deficits in oral sensation have longer delays in the onset of laryngeal ascent and consequently an increased aspiration risk.

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NEUROLOGICAL PICTURE

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Chiari I malformation with holocord syrinx

A 23-year-old man presented with a 1-month history of headache and numbness of the left arm and hand. Neurological examination showed hypesthesia in the left arm. Magnetic resonance imaging of the spine showed peg-shaped herniation of tonsils 9 mm below the foramen magnum (fig 1A), with altered signal intensity in the medulla (fig 1B). There was no hydrocephalus. T1W and T2W screening of the whole spine showed holocord syringohydromyelia (fig 2A,B). No focal intraspinal mass was seen. Chiari I malformation with a holocord syrinx was diagnosed.

Chiari I malformation is traditionally defined as inferior displacement of the cerebellar tonsils and, sometimes, the inferior vermis through the foramen magnum into the rostral cervical canal. Between the ages of 5 and 15 years, descent of upto 6 mm is not considered pathological. However, in older patients, tonsillar protrusion $\geqslant$5 mm is associated with a marked rise in the onset of clinical symptoms.[1]

The aetiopathogenesis has been a focus of considerable controversy. It may be congenital or acquired. The causative factors include underdevelopment of the posterior fossa, malformation of the craniocervical junction, and changes in intracranial pressure.

The clinical presentation is varied. The patients may present with headache or oculomotor disturbances (blurred vision, oscillopsia). Otoneurological complaints (dizziness, tinnitus), cranial neuropathies (dysphagia, dysarthria) or cerebellar abnormalities (dysmetria, tremor) may be the other modes of presentation. Potentially serious autonomic disturbances are also common: sleep apnoea, respiratory failure, syncope and even sudden death. Skeletal anomalies that may be seen in association with Chiari I malformation include basilar impression, atlanto-occipital fusion, atlanto-axial assimilation, Klippel Feil deformity and scoliosis.[2 3]

The concurrence of Chiari I malformation and syringohydromyelia has been estimated at between 25% and 65%. This results from obstruction of cerebrospinal fluid circulation in the cisterna magna. The presence of a syrinx is more commonly associated with symptoms.[3]

The various surgical treatment options can be broadly categorised into four groups: (1) decompression of hindbrain malformation by suboccipital craniectomy and upper cervical laminectomy; (2) laminectomy and syringostomy; (3) terminal ventriculostomy; and (4) percutaneous aspiration of the syrinx. The first group is the most common surgical option used in cases of Chiari I malformation and syringohydromyelia.[2]

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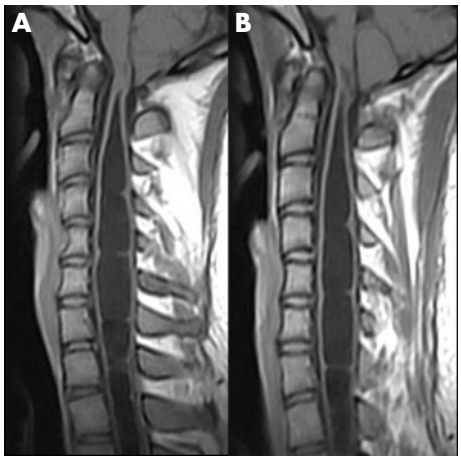

Figure 1 (A,B) Consecutive sagittal T1W images show herniation of peg-shaped cerebellar tonsils 9 mm below the level of the foramen magnum. The visualised cervical cord shows mild expansion with hydrosyringomyelia. The medulla shows altered signal intensity, distinct from the cord syrinx, probably due to pressure necrosis.

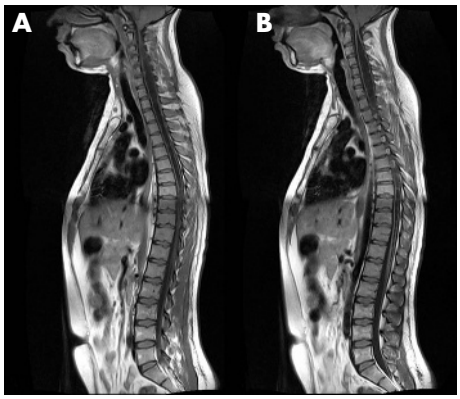

Figure 2 (A,B) Sagittal T1-weighted image of the whole spine shows the syrinx involving the entire cord.

Competing interests: None declared.

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